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THE CONSTRUCT VALIDITY OF PTSD AND DEPRESSION IN THE CONTEXT OF

TRAUMA THROUGH AN EXAMINATION OF EMOTION REGULATION

STRATEGIES

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Abstract

Posttraumatic stress disorder (PTSD) and depression overlap functionally as well as in terms of symptomatologies. Within the context of trauma, this overlap makes it difficult to determine whether depression is a distinct construct from PTSD. Through an examination of specific strategies used to regulate emotions, the nature of the functional overlap between the two disorders was investigated. Specific emotion regulation strategies were examined in a mixed clinical and community sample of 64 couples in which at least one partner screened positive for PTSD. Factor analyses of PTSD and depression measures were conducted in order to further clarify the symptom structure of the disorders. Using bivariate correlations and comparisons of the strength of the correlations, relations between specific emotion regulation strategies and symptoms clusters of PTSD and depression were then examined in order to potentially differentiate PTSD and depression within the context of trauma. Results suggest that PTSD and depression within the context of trauma are not distinct constructs. Implications are further discussed.

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Chapter 1

INTRODUCTION

Posttraumatic stress disorder (PTSD) is unique amongst psychological disorders in that its development requires a specific etiological incident that evokes fear, helplessness, or horror in an individual (APA, 2000). While most people have experienced this sort of traumatic event (i.e., estimates range from 51% to 90%; Breslau & Davis, 1992; Kessler et al., 1995), only a subset of these individuals develops PTSD. Specifically, the lifetime prevalence rate of PTSD is estimated to be 6.8% in the US population (Kessler, Chiu, Demler & Walters, 2005). Although PTSD is commonly conceptualized using a categorical diagnostic approach, it is widely accepted that PTSD is best measured dimensionally, as indicated by a series of taxometric analyses as well as theoretical conceptualizations of the natural recovery process following trauma (Asmundson et al., 2000; Broman-Fulks et al., 2006; Forbes, Haslam, Williams & Creamer, 2005; Ruscio, Ruscio & Keane, 2002).

Overlap Between PTSD and Depression

PTSD has an extremely high comorbidity rate with other psychological disorders. While there is a high prevalence of comorbidity with substance use disorders, mood disorders, and other anxiety disorders, Major Depressive Disorder (MDD) has the highest comorbidity rate (Breslau & Davis, 1992). Due to the high rates of comorbid MDD among individuals with PTSD, there has been debate regarding whether MDD in the context of trauma is a separate construct from PTSD, or if the two comprise the same construct. Given the large overlap between the disorders, it is important to examine the question of the construct validity of PTSD and MDD within the context of trauma. Alternate conceptualizations of PTSD have implications for future research and directions of the field in terms of challenging the current

conceptualization of PTSD as an anxiety disorder with behavioral avoidance at its core. It is also important in order to gain more information to maximize prevention and treatment response efforts in regard to trauma and to ensure that all important aspects of a traumatized individual's experience are being assessed and addressed quickly and effectively.

Shared Constructs Argument

There are many reasons why PTSD and depression appear to be indistinguishable constructs. Supporters of the shared constructs argument have presented findings indicating that MDD increases the risk of an individual being exposed to a PTSD-inducing traumatic event (Breslau, Davis, Peterson & Schultz, 1997; Bromet, Sonnega & Kessler, 1998; Connor & Davidson, 1997), PTSD increases the likelihood that an individual will have his/her first onset of MDD (Breslau et al., 1997; Kessler et al., 1995), and an elevated risk for MDD exits in individuals with PTSD, but not in trauma-exposed individuals without PTSD (Breslau, Davis, Peterson & Schultz, 2000). Related to this, Schindel-Allon and colleagues (Schindel-Allon, Aderka, Shahar, Stein & Gilboa-Schechtman, 2010) found evidence that depressive symptoms increase the severity of PTSD symptoms over time and PTSD and depression symptoms are synchronously connected (i.e., significantly correlated) immediately post-trauma (i.e., 24-48 hours), and two, four, and twelve weeks post-trauma. (Although evidence exists suggesting that anxiety symptoms increase the severity of depression symptoms [Bromberger & Matthews, 1996; Cole, Peeke, Martin, Truglio & Seroczynski, 1998; Wetherell, Gatz & Pederson, 2001], this was not found in Schindel-Allon and colleagues' study.) These dual relationships suggest that PTSD and depression change together and partially originate from mutual risk factors (Schindel-Allon et al., 2010), which provides support for a shared constructs argument.

Examining the issue of symptom overlap is key in determining the separability of the constructs of PTSD and MDD, as this has been proposed to account for a large part of the comorbidity between the two disorders (Keane, Taylor & Penk, 1997; Shalev et al., 1998, Brady, Killeen, Brewerton & Lucerini, 2000). The symptoms of anhedonia, diminished concentration, and sleep problems are present in both disorders, and therefore have been hypothesized to be "contaminated" symptoms (Franklin & Zimmerman, 2001). Franklin and Zimmerman (2001) tested this contamination hypothesis and found that the contaminated symptoms were not less specific to PTSD, were not less correlated with PTSD total score, and were not differentially endorsed by PTSD individuals with and without depression. This suggests that symptom overlap is not responsible for the high comorbidity between PTSD and MDD. Taft and colleagues (Taft, Resick, Watkins & Panuzio, 2009) also found that the relationship between PTSD and MDD does not appear to be due to symptom overlap through examining rates of depression in a PTSD sample with the full Beck Depression Inventory (BDI) versus the BDI with the three overlapping items removed. They found that the confidence intervals for the two rates overlapped, which led to the conclusion that the two disorders in the context of PTSD were not considerably different from each other. Elhai and colleagues (Elhai, Carvalho, Miguel & Primi, 2010; Elhai, Grubaugh, Kashdan & Frueh, 2008) also concluded that symptom overlap is not the cause of the comorbidity between the disorders, after examining PTSD diagnostic status from the National Comorbidity Survey Replication (NCS-R) with and without the overlapping MDD symptoms and finding no significant difference in PTSD diagnostic prevalence. They further concluded that PTSD and MDD represent a single, underlying dimension (Elhai et al., 2010). Grubaugh and colleagues (Grubaugh, Long, Elhai, Frueh & Magruder, 2010) echoed this conclusion through examining diagnostic PTSD status with and without the symptoms that overlap with MDD. They

also concluded that removing the overlapping symptoms did not affect PTSD prevalence, comorbidity status, functional impairment, or structural validity (Grubaugh et al., 2010).

Aside from shared symptoms, there are other factors at play in the PTSD-MDD overlap. There are several cognitive mechanisms that are shared between PTSD and depression (e.g., increased access to negative memories and decreased access to positive memories, similar attributional styles; Hyer, Stanger & Boudewyns, 1999; Joseph, Williams & Yule, 1995; McNally, Kaspi, Reimann & Zeitlin, 1990), as well as negative affectivity (Schindel-Allon et al., 2010) and impaired emotional processing (Hyer et al., 1999; Rachman, 1980). Additionally, several risk factors are common to both PTSD and depression, such as female gender, a history of child abuse, and a prior history of depression (Breslau et al., 1998; Carlson & Rosser-Hogan, 1991; Kendler, Gardner & Prescott, 2002). Another important factor to consider is the influence of genetics, which has been found to partially account for the association between PTSD and MDD (Fu et al., 2007; Koenen et al., 2008). Several genes have been associated with PTSD (Koenen, Nugent & Amstadter, 2008), and among these, multiple genes are also associated with depression (i.e., FKBP5, 5-HTT, serotonin transporter promoter; Binder et al., 2004; Caspi et al., 2003; Lee et al., 2005). It has also been found that the genetic influences on depression account for the majority of the variance in PTSD (Koenen et al., 2008; Fu et al., 2007). Though there is much that is unknown regarding genetics in psychopathology, this work provides preliminary support for a lack of discriminant validity from a biological perspective.

Distinct Constructs Argument

Conversely, the body of research that supports a distinct constructs argument suggests that there is a sequential, chronological onset of the two disorders, with depression following PTSD and acting as a "secondary" disorder (Shalev et al., 1998). Three studies have been

conducted that support the distinct pathways model. In the first study, Shalev and colleagues (1998) studied injury survivors recruited immediately post-trauma in the emergency room, then assessed one week, one month, four months, and one year post-trauma. Compared to symptoms of depression, symptoms of PTSD were found to be more strongly and specifically associated with early autonomic activation measured in the emergency room immediately post-trauma, insomnia, intrusion, and auditory startle. Thus, while acknowledging that there are many shared risk factors, and often PTSD and depression develop concurrently in the aftermath of trauma, Shalev and colleagues (1998) concluded that PTSD and depression develop separately (i.e., it is possible for the disorders to develop at the same time, yet exist as separate entities that have different etiologies). In the second study, O'Donnell and colleagues (O'Donnell, Creamer & Pattison, 2004) found that while PTSD and depression following trauma are indistinguishable in the long term (i.e., 12 months following the traumatic event), in the three months following the event, PTSD and depression are distinct and independent (i.e., developing concurrently but separately), due to different patterns of pathology at the different time points. These differences between patterns of pathology are highlighted by the high degree of motion between diagnostic categories, for example high rates of individuals with PTSD only at the first time point and MDD only at the second time point, and vice versa. In the third study, Blanchard and colleagues (Blanchard, Buckley, Hickling & Taylor, 1998) concluded through multivariate structural equation modeling that PTSD and depression represented separate constructs, however the two constructs correlated at .88, which reduces the strength of their argument.

Conclusions to be drawn

Although the question of shared versus distinct etiology is not resolved and there is still much that is not known about the construct validity of both disorders, the evidence points more

heavily in favor of the shared constructs argument. The research in support of the shared

constructs argument is consistent and more cohesive than the distinct constructs argument, which

has conflicting evidence. O'Donnell and colleagues (2004), who posit that the disorders develop

separately immediately post-trauma, also hypothesize that the disorders become the same

construct with time (which shows partial support for the shared constructs argument). Shalev and

colleagues (1998) conjecture that different factors predict PTSD and MDD, but also state that the

disorders often develop concurrently (i.e., they develop at the same time but are predicted by

different factors and stay separate over time). This contradicts the work of O'Donnell and

colleagues (who posit that the disorders start out separately and becoming one construct over

time). Overall, the shared constructs argument appears to be more valid than the distinct

constructs argument. The shared constructs argument should continue to be tested, however, to

validate PTSD/MDD as a construct within the context of trauma.

Factor Structure of PTSD and Depression

One method of gaining clarity on this issue is to consider the unique structures of PTSD

and depression in order to examine the ways in which some aspects of these disorders may

overlap while other aspects may be distinct. Having a clearer picture of the latent variables that

make up the core of each disorder may inform whether the core of these disorders is similar or

distinct.

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text

Revision (DSM-IV-TR; APA, 2000) describes PTSD as being comprised of three symptom

clusters: reexperiencing (i.e., upsetting thoughts or memories of the trauma, nightmares, reliving

the trauma as a flashback, distress in response to trauma cues, physiological reactivity to trauma

cues), avoidance (i.e., avoiding thoughts, feelings, or conversations associated with the trauma,

avoiding activities, places, or people associated with the trauma, inability to remember aspects of the trauma, diminished interest in previously enjoyed activities, feeling detached from others, restricted range of affect, sense of foreshortened future), and hyperarousal (i.e., exaggerated startle, hypervigilance, sleep difficulty, irritability or anger outbursts, difficulty concentrating). However, there has been considerable support for a four-factor model in place of the three-factor model, in which the avoidance cluster is broken into two separate clusters as proposed by Foa and colleagues (Foa, Zinbarg & Rothbaum, 1992): Behavioral avoidance (i.e., avoiding thoughts, feelings or conversations associated with the trauma, avoiding activities, places, or people associated with the trauma, a sense of foreshortened future, and the inability to remember aspects of the trauma) and emotional numbing (i.e., diminished interest in significant activities, feeling detached from others, and a restricted range of affect). The majority of research conducted on this issue supports the four-factor model (Blake et al., 1990; Elklit & Shevlin, 2007; King, Leskin, King & Weathers, 1998; McWilliams, Cox, & Asmundson, 2005; Naifeh, Elhai, Kashdan & Grubaugh, 2008; Scher, McCreary, Asmundson & Resick, 2008). While there has been some support for a non-traditional four-factor model (McWilliams et al., 2005), there has been a paucity of work that supports the three-factor model (i.e., Cox, Mota, Clara & Asmundson, 2008; supported both three-factor and four-factor models).

Although there is no standard structure for symptom clusters in depression as there is in PTSD, a general pattern has emerged. Several studies have examined the structure of depressive symptoms using the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erbaugh, 1961), one of the most widely used depression assessment measures in the field. Dunn and colleagues (Dunn et al., 2002) found a three-factor structure, including clusters of negative cognitions, psychomotor-anhedonia symptoms, and vegetative symptoms. Grunebaum and

colleagues (Grunebaum et al., 2005) found a similar pattern; however, they labeled their clusters self-blame, subjective depression, and somatic complaints. Dunn and colleagues' negative cognitions cluster shares many similar aspects with Grunebaum and colleagues' self-blame cluster (i.e., sense of failure, feelings of punishment, self-blame, self-criticism), while Dunn and colleagues' psychomotor-anhedonia cluster shares many aspects of Grunebaum and colleagues' subjective depression cluster (i.e., indecisiveness, difficulty getting work done, lack of satisfaction). Dunn and colleagues' final cluster of vegetative symptoms is also quite similar to Grunebaum and colleagues' somatic complaints cluster (i.e., decreased appetite, disturbed sleep, weight loss). Kashdan and colleagues (Kashdan, Elhai & Frueh, 2006), however, found a two-factor solution including anhedonia and undifferentiated global depressive symptoms. The fact that Kashdan and colleagues used a PTSD sample is noteworthy, and highlights the importance of anhedonia in PTSD, as these specific symptoms were present in a trauma-exposed sample (as opposed to the more vague nature of the remaining symptoms which did not cohesively covary). The non-specific nature of the two-factor model (i.e., undifferentiated global depressive symptoms) combined with the general agreement of the other researchers on the three-factor model provides stronger support for a three-factor model of depressive symptoms. Given the findings of Kashdan and colleagues (2006), in terms of the importance of anhedonia in individuals who have experienced traumatic events, it is also important to further examine this issue in a trauma sample.

While many studies examining the overlap between PTSD and MDD have focused on the categorical DSM-IV diagnosis of MDD, there is a sizeable body of research indicating that depression is best conceptualized by a dimensional, rather than categorical model. This argument has been supported by taxometric analyses in both community and clinical samples (Flett,

Vrendenburg & Krames, 1997; Slade, 2007; Wynn, 2007). A dimensional model of depression is consistent with findings that PTSD is best represented dimensionally (Asmundson et al., 2000; Broman-Fulks et al., 2006; Forbes, et al., 2005; Ruscio et al., 2002), and allows for the consideration of individual variability among symptom clusters that have been derived from a dimensional model using the BDI. Therefore, a dimensional model will be used in the current study in order to examine depressive symptoms and PTSD symptoms instead of focusing on the categorical diagnoses of MDD and PTSD. The factor structure of depression and PTSD will be examined in a trauma sample in order to also clarify the divergent findings in this area.

The Importance of Emotion Regulation

A second method of gaining clarity on the issue of if depression is a distinct construct from PTSD in the context of trauma is to examine possible underlying mechanisms that give insight into differential functioning of PTSD and depressive symptoms. Emotion regulation deficits are present across numerous forms of psychopathology, and are salient in both PTSD and depression (e.g., Ehring, Fischer, Shnülle, Bösterling & Tuschen-Caffier, 2008; Liverant, Brown, Barlow & Roemer, 2008; Tull, Barrett, McMillan & Roemer, 2007a; Tull, Jakupcak, Paulson & Gratz, 2007b). These deficits may be seen as a pretrauma risk factor (i.e., existing deficits in emotion regulation make an individual more likely to develop PTSD following a potentially traumatic event) or as a mechanism (i.e., an individual has no pretrauma emotion regulation deficits, but after experiencing a traumatic event is not able to use emotion regulation effectively and begins using maladaptive emotion regulation strategies chronically, which acts as a causal factor for PTSD). While it is not currently possible to make the differentiation between risk factor or mechanism, emotion regulation is salient in some capacity. As will be discussed below, several reasons exist for proposing that different forms of emotion regulation could be

differentially related to PTSD and depression. On a broad level, given the prevalence of emotion regulation deficits in both PTSD and depression, it is logical to pursue emotion regulation as a possible mechanism to potentially differentiate the constructs. If distinct forms of emotion regulation deficits are differentially associated with PTSD versus depressive symptoms, the theory that PTSD and depression within the context of trauma are similar constructs may begin to be falsified.

Emotion regulation is a hot topic in psychology research, yet there is no cohesive, generally accepted definition. There are various, related, frameworks of emotion regulation that highlight different aspects (e.g., cognitive, emotional control), but no overarching connective theory. The nebulous state of emotion regulation research highlights the importance of clear operationalization of constructs. For the purposes of the current study, emotion regulation will be defined in a broad sense, as processes in an individual that modify an activated emotion (Cole, Martin & Dennis, 2004). These processes involve either changes in the emotion itself (e.g., changes in duration and/or intensity; Thompson, 1994) or changes in other processes, such as social interaction, behavior, cognition, or memory, which in turn affect the activated emotion (Cole et al., 2004). Emotion regulation can be both adaptive and maladaptive, the latter being commonly seen in psychopathology and also prominent in both PTSD and depression. Maladaptive emotion regulation is defined in this context as an individual using an emotion regulation strategy that furthers his/her psychopathology or the course of the disorder. The individual may see maladaptive emotion regulation as adaptive, as it may relieve distress in the moment, but it is maladaptive in the long term. Even though emotion regulation is a complex entity, Cole and colleagues' (2004) definition provides a good working foundation on which to further build.

Model of Emotion Regulation Used in the Current Study

While operationalizing the construct is a key first step, it is also necessary to establish a larger framework in which to understand emotion regulation (see Figure 1). Gross and John (Gross, 1998; Gross, 2002; Gross & John, 2003; John & Gross, 2004) have been instrumental in shaping the field of emotion regulation, particularly with their process model of emotion regulation (Gross, 2002; Gross & John, 2003; Gross & Thompson, 2007; Werner & Gross, 2010). In this model, emotion regulation is viewed as an ongoing process, in which an emotion cue triggers a series of responses, or regulatory strategies, that occur over time. This builds on Cole and colleagues' (2004) definition of changes in activated emotions or other processes by adding consideration of a chronological order (i.e., either antecedent-focused, before the emotion has a chance to fully activate, or response-focused, after an emotion has been activated and a preliminary response occurs). Within this process model, different emotion regulation strategies have different consequences. The focus of the original model was on cognitive reappraisal (antecedent focused, where an individual "rethinks" the situation and interprets it in a different manner than the previous interpretation) and expressive suppression (response focused, where an individual restricts an expression of an emotional response after the dysregulating stimulus has occurred) as the focal emotion regulation strategies (Gross, 2002). However, many other strategies fit into the model. Gross and colleagues posit that adaptive emotion regulation is multifaceted, and other strategies can be incorporated into this process model (Gross & Thompson, 2007; Werner & Gross, 2010). Here I discuss how clearly defined and commonly examined emotion regulation strategies fit into this expanded process model.

The first point of the expanded process model is *situation selection*, where the individual makes the choice to enter a situation that may elicit emotions. An important action at this step is

not reacting to either the dysregulating situation or one's own internal reaction instantly, but instead taking a moment before reacting. Werner and Gross (2010) posit that this allows the emotion to emerge without avoidance, resistance, or impulsive behaviors. Impulse control difficulties can become an obstacle here, as individuals with impulse control difficulties are less able to take this pause. Such individuals will not make conscious choices regarding emotion regulation; instead, they will rely on natural impulse. Another important emotion regulation strategy relevant at this point is being aware of long-term goals and the ability to resolve whether possible courses of action are consistent with those goals (Werner & Gross, 2010). Keeping one's goals in mind and planning for goal-consistent behavior is an early step, similar to impulse control, and occurs even before the individual decides to enter into a potentially emotion-eliciting situation (e.g., an individual with difficulties engaging in goal-directed behavior will have trouble gauging whether entering into a particular situation will be consistent with and/or beneficial for his/her goals). Looking at the idea of pausing so as not to immediately react, avoidance can be an important issue around this time, specifically in terms of emotional avoidance. If an individual refuses to accept that s/he may be about to experience an emotion, consciously or not, it becomes impossible to take this pause to regulate. Thus, nonacceptance of emotion occurs very early in the emotion regulation process model, even before the point of situation selection. If one is refuses to accept that s/he is about to experience an emotion, it is not possible to begin the process of emotion regulation.

Thompson and Gross' (2007) next point is *situation modification*, where the individual takes steps to alter the emotional impact of the situation. Situation modification can be adaptive, as in telling jokes in a social situation, but can also be maladaptive, as in the case of an individual with obsessive compulsive disorder using a public restroom, yet using paper towels in

order to avoid actually touching anything (Werner & Gross, 2010). Situation modification is followed by *attentional deployment*, where the individual selects which aspects of the situation to focus on in order to influence his/her emotions. Unfortunately, these two points are rarely empirically examined in relation to specific emotion regulation strategies.

The next point in Gross and Thompson's (2007) model is *cognitive change*, where the individual changes his/her interpretation of the situation's meaning (i.e., cognitive reappraisal). This is done through thinking about how the individual can handle the requirements of the situation (Werner & Gross, 2010). After this point in the process model, an emotion is expected to occur.

Once an emotion occurs, adaptive emotion regulation requires knowing that one is experiencing an emotional response and being able to identify the emotion being experienced (Werner & Gross, 2010). Gratz and Roemer (2004) identify these strategies as lack of emotional awareness (i.e., an individual not knowing when s/he is experiencing any type of emotion) and lack of emotional clarity (i.e., knowing an emotion is being experienced, but not being able to identify which emotion is it). Since knowledge of an emotional response must occur before knowing what the emotional response is, these two strategies are chronologically linked. Both of these strategies occur early in the response-focused stage of the process model.

The final point in Gross and Thompson's model (2007) is *response modulation*, where the individual tries to influence his/her emotional responses after the dysregulating stimulus/situation occurs. Expressive suppression occurs during the response modulation step, as the individual is restricting his/her response post-dysregulating event. In an attempt to control what the individual perceives to be a potentially intolerable emotional experience, s/he focuses on pushing the emotional expression away. Another emotion regulation strategy that fits in at the

point of response modulation is rumination. Rumination can be explained through the response styles theory (Nolen-Hoeksema, 1991), which suggests that rumination is a style of responding to distress that includes passively and repetitively focusing on the symptoms of such distress and the possible origins of the symptoms, as well as the possible future consequences of the symptoms. The key aspect of rumination is perseverative thinking, which is associated with several negative cognitive styles (Lyubomirsky & Nolen-Hoeksema, 1995; Nolen-Hoeksema & Davis, 1999). Nolen-Hoeksema explains rumination as a passive process. Since rumination is past-focused, it by definition occurs after the emotion has occurred, when individuals attempt to change their feelings by repetitively thinking about the dysregulating stimulus.

Throughout the different points of the process model, adaptive emotion regulation requires flexibility in terms of modifying responses in order to achieve longer term goals (Barrett & Gross, 2001; Linehan, 1993), and selecting appropriate strategies in order to successfully regulate one's emotions (Werner & Gross, 2010; Berenbaum, Raghavan, Le, Vernon & Gomez, 2003; John & Gross, 2004; Kring & Werner, 2004; Mennin & Farach, 2007). Gratz and Roemer (2004) highlight the importance of flexibility of emotion regulation strategies in adaptive emotion regulation. Lack of flexibility of emotion regulation strategies is relevant throughout the process of emotion regulation, and occurs throughout the five points in the model.

Emotion Regulation in PTSD

PTSD develops following a strong emotional response (i.e., fear, helplessness, or horror; APA, 1994) to a potentially traumatic event. PTSD is then advanced through avoidance of the emotional response evoked by memories of the trauma, which in turn leads to more reexperiencing symptoms and related emotions that accompany such symptoms. More specifically, behavioral avoidance and emotional numbing lead to higher reexperiencing

symptoms (i.e., by trying to avoid them, the distressing reminders and other reexperiencing symptoms actually increase), which in turn leads to increased avoidance and emotion numbing (i.e., to try even harder to get rid of the distressing reminders; Hayes et al., 2004; Litz, 1992). The reexperiencing symptoms cause the individual to be constantly on guard or alert, which is at the core of the hyperarousal symptom cluster (Post, Weiss & Smith, 1995). Although much is not known about why people with PTSD fail to exhibit natural recovery after a traumatic event, one factor that may play a significant role is the use of maladaptive emotion regulation strategies (Tull et al., 2007a).

Previous research supports the idea that emotion regulation difficulties are significantly correlated with PTSD symptoms (Ehring & Quack, 2010; Forster, Schoenfeld, Marmar & Lang, 1995; Roemer, Litz, Orsillo & Wagner, 2001; Steiner, Garcia & Matthews, 1997; Tull et al., 2007a; Tull et al., 2007b; Wolf, Alavi & Mosnaim, 1988). Tull and colleagues (2007a) found that nonacceptance of emotions, a strategy occurring in the early stages of the Gross and Thompson's (2007) process model, was uniquely associated with posttraumatic stress symptoms, above and beyond shared variance with negative affect. PTSD has also been shown to be associated with the strategic withholding of emotions (Roemer et al., 2001), which is similar to nonacceptance of one's emotions. These aspects of being unwilling to accept emotions are considered to be an avoidance strategy, specifically avoidance of emotional responses associated with trauma reminders in PTSD. Through this process, the individual is able to avoid the emotion and to remain anesthetized to feeling anything. Thus, in the current study, nonacceptance of emotion is expected to correlate highly with PTSD symptoms, particularly the emotional numbing symptom cluster.

As individuals with PTSD face potentially dysregulating situations, it may be difficult for them to make adaptive choices. Impulse control deficits, which also occur in the early stages of the process model (Gross & Thompson, 2007), have been consistently found in individuals with PTSD (Forster et al., 1995; Steiner, Garcia & Matthews, 1997; Wolf et al., 1988). Impulse control issues have been linked with anger (Novaco, 2010), which is a symptom in the hyperarousal cluster of PTSD. Impulsivity has, in fact, been conceptualized as a symptom of hyperarousal in PTSD (Weiss, 2007). In addition, Tull and colleagues (2007a) found that difficulties with impulse control were uniquely associated with posttraumatic stress symptoms, above and beyond shared variance with negative affect. Difficulties with impulse control are, therefore, expected to correlate highly with PTSD, particularly the hyperarousal symptom cluster.

Emotion Regulation in Depression

Emotion dysregulation has been shown to be more severe among individuals with depression than healthy controls (Ehring et al., 2008; Liverant et al., 2008; Wei & Fu, 2008). Prior research suggests that there are several salient emotion regulation strategies to consider in reference to depression. Unlike the proposed emotion regulation deficits in PTSD, which occur in the early stages of the process model, the proposed emotion regulation deficits in depression largely occur in the later stages of the model.

If an individual with elevated depressive symptoms experiences sad mood and diminished affect, in addition to the fatigue symptoms typical of depressive episodes, energy and motivation may be low (Frank et al., 2007), likely contributing to the decreased engagement in significant activities that is typical of depressive episodes (APA, 2000). Thus, individuals with elevated depressive symptoms experience difficulties completing goals (Haeffel, Abramson,

Brazy & Shah, 2008), potentially as a means of coping such that abandoning the pursuit of goals may be easier than facing the possibility of failing to accomplish such goals. Therefore, it may be a coping strategy, although maladaptive, to avoid engaging in goal-directed behavior. Thus, expectations of failure and hopelessness are elevated in depression (Haeffel et al., 2008). This lack of motivation or desire to accomplish things is consistent with the symptoms hypothesized to comprise the vegetative symptom cluster (e.g., lack of energy, being tired/fatigued). In the current study, the ability to engage in goal-directed behavior is proposed to be highly correlated with depression, particularly the vegetative symptom cluster. The ability to engage in goal directed behavior occurs early in Gross and Thompson's (2007) process model, and is the only strategy hypothesized to correlate highly with depression that occurs in the early stages of the model.

Lack of emotional awareness is another emotion regulation deficit that has been found to be associated with depressive symptoms (Hatzenbuehler, McLaughlin & Nolen-Hoeksema, 2008), potentially developing from persistent sadness and rumination (Rieffe, Oosterveld, Miers, Terwogt & Ly, 2008). This lack of emotional awareness is likely associated with other symptoms of anhedonia, due to the lack of recognition of emotion being experienced not permitting the individual to gain pleasure from enjoyable activities. In addition, if an individual is having trouble distinguishing if s/he is experiencing an emotion, it would be more difficult to identify the emotion. This lack of emotional clarity has been previously found in depressed individuals (Ehring et al., 2008; Rude & McCarthy, 2003). Both lack of emotional awareness (i.e., when an individual is unaware that s/he is experiencing en emotion) and lack of emotional clarity (i.e., when an individual is aware that s/he is experiencing an emotion, but cannot identify which emotion is being experienced) likely either lead to, or are a consequence of, anhedonia in

depression. These strategies both occur during the later stages of Gross and Thompson's (2007) process model, and occur before expressive suppression (i.e., when an individual suppresses an emotional response). Similar to lack of emotional awareness, the refusal of an individual to express the emotion s/he is feeling may contribute to or be a product of anhedonia. Expressive suppression occurs at a later point in the model than lack of emotional awareness, but cannot occur in conjunction with it, as an individual cannot suppress an emotional expression is s/he is unaware than an emotion is being experienced. Therefore, these three strategies (i.e., lack of emotional awareness, lack of emotional clarity, and expressive suppression) are expected to correlate highly with depression, particularly the anhedonia symptom cluster.

Another important strategy when considering emotion regulation deficits in depression is rumination, a strategy at the latest stage in the process model (i.e., response modulation, Gross & Thompson, 2007). Rumination has been strongly and consistently linked with depression (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema, Wisco & Lyubomirsky, 2008), and is expected to demonstrate a strong association with depressive symptoms, particularly the cognitive symptom cluster due to the high cognitive demand of repetitive thinking.

Emotion Regulation Strategies Common to both PTSD and Depression

As reviewed, PTSD has primarily been associated with emotion regulation deficits in the early stages of the process model, while depression has been primarily associated with deficits in the middle to late stages of the model (Gross & Thompson, 2007). Cognitive reappraisal, a generally adaptive emotion regulation strategy according to Gross and John (2003), is likely important to both depression and PTSD, and occurs in the later stages of the process model. Cognitive reappraisal can be used to decrease maladaptive thinking patterns that are common in

depression, and when it is used, is often used as an adaptive strategy. In addition, reappraisal of potential threat cues may be extremely helpful in PTSD in terms of realizing that something originally identified as a threat is not actually dangerous (e.g., thinking discarded trash by the side of the road is a roadside bomb, but upon reappraising, realizing that it is just trash). Given the depth of dysregulation in both depression and PTSD, reappraisal is likely an underused strategy. Therefore, diminished use of cognitive reappraisal is expected to correlate similarly in strength with both PTSD and depression symptoms, thus not serving to differentiate the two constructs.

Another important aspect of emotion regulation to consider in both PTSD and depression is the flexibility of an individual to access emotion regulation strategies. In regards to depression, Bonanno and colleagues (Bonnano, Papa, Lalande, Westphal & Coifman, 2004) have suggested that this flexibility is key in order for an individual to adjust in a healthy manner in response to potentially dysregulating stimuli. Tull and colleagues (2007a) also found that limited access to emotion regulation strategies was uniquely associated with posttraumatic stress symptoms. This flexibility occurs throughout Gross and Thompson's (2007) process model. If an individual only has one strategy available to him/her and it is not appropriate for a particular situation, emotional distress and increased severity of PTSD and/or depressive symptoms will be experienced, as the individual is unable to regulate emotions successfully. Thus, lack of flexibility of emotion regulation strategies is also expected to correlate similarly in strength with both PTSD and depression, thus not serving to differentiate the two constructs.

Current Study

In the current study, measures of self-reported emotion regulation strategies were correlated with depression and PTSD symptom clusters in order to examine whether different

emotion regulation strategies are more strongly associated with PTSD or depressive symptoms. The goal of this examination is to be able to better understand the functional overlap between the two disorders, and to give insight into whether they are one shared construct or two distinct constructs. It is also a goal to gain a better understanding of the structure of PTSD and depression, and the emotion regulation strategies that may be more strongly associated with each symptom cluster.

If PTSD and depression in the context of trauma are similar/equivalent constructs, emotion regulation will not differentiate between the two disorders. If PTSD and depression are two separate constructs, however, differences in emotion regulation strategies across the two disorders should be evident. If PTSD and depression are, in fact, two separate disorders, the differences should emerge in the following domains.

(a) Nonacceptance of emotions and difficulties with impulse control will correlate more highly with PTSD than depressive symptoms.

(a1) Nonacceptance of emotion will correlate more highly with the emotional numbing symptom cluster than with the depression total score or any of the depression symptom clusters (i.e., vegetative, anhedonia, and cognitive).

(a2) Difficulties with impulse control will correlate more highly with the hyperarousal symptom cluster than with the depression total score or any of the depression symptom clusters.

(b) Difficulties with goal-directed behavior, lack of emotional awareness, lack of emotional clarity, expressive suppression, and rumination will correlate more highly with depressive symptoms than PTSD symptoms.

(b1) Difficulties with goal-directed behavior will correlate more highly with the

vegetative symptom cluster than with the PTSD total score or any of the PTSD symptom clusters (i.e., reexperiencing, behavioral avoidance, emotional numbing, hyperarousal).

(b2) Lack of emotional awareness will correlate more highly with the anhedonia symptom cluster than with the PTSD total score or any of the PTSD symptom clusters.

(b3) Lack of emotional clarity will correlate more highly with the anhedonia symptom cluster than with the PTSD total score or any of the PTSD symptom clusters.

(b4) Expressive suppression will correlate more highly with the anhedonia symptom cluster than with the PTSD total score or any of the PTSD symptom clusters.

(b5) Rumination (brooding and reflection) will correlate more highly with the cognitive cluster than with the total PTSD score or any of the PTSD symptom clusters

(c) Cognitive reappraisal and lack of flexibility of emotion regulation strategies will not differentially correlate between PTSD and depression total scores or any symptom clusters.

Chapter 2

METHOD

Participants

Participants included 128 individuals from 64 couples in which at least one partner screened positive for probable PTSD. Couples were heterosexual, either married or living together as a romantic couple, and between the ages of 18 and 65. Because a dimensional approach was used to conceptualize PTSD and depressive symptoms, both partners in each couple were included in the analyses in order to best capture the full range of experiences. Of the couples who contacted the lab about the study ($n = 276$), 201 couples completed the initial screening. Of these, 67 couples were invited to participate, and the remaining couples were not invited to participate due to neither partner meeting screening criteria for probable PTSD ($n = 122$), partners not being interested following completion of the telephone screen ($n = 8$), partners' combined income exceeding $100,000 per year and/or either partner had more than six years post-high school education ($n = 3$), or partners ending their relationship ($n = 1$). The participants who took part in the study had an average age of 37 (range of 18-64, $SD = 12.64$). The sample was 84% Caucasian ($n = 108$), 6% African American ($n = 7$), 4% biracial/multiracial ($n = 5$), 4% Hispanic or Latino ($n = 5$), and 2% not reported ($n = 3$). The average monthly income per individual was $1,708.10.

Procedure

Participants were recruited from rural communities in central Pennsylvania through local newspapers, online advertisements, and posted flyers (posted at local businesses, medical offices, and community centers), and two outpatient psychological clinics. Six couples were recruited through the clinics; 269 were recruited through the community. Each partner in the couple was

independently screened over the telephone, at which time the study was explained and demographic information was gathered. In addition, Criterion A status was assessed, and a PTSD screener (the Posttraumatic Checklist; see below) was administered to both partners, with each partner screened separately. Both partners had to be between the ages of 18 and 65, and able to speak and write in English. Both partners had to be married or living together in a heterosexual relationship, and at least one partner must have experienced a Criterion A traumatic event and meet the cut point criteria on the Posttraumatic Checklist described below. After eligibility was determined by the Principal Investigator, couples came to the lab for either two four-hour sessions or one eight-hour session. The data used for the present analyses came from this larger study. Individuals were assessed for PTSD with a clinical interview administered by a clinical psychology graduate student. Upon completion of the lab portion of the study, participants were asked to complete several paper and pencil questionnaires at home, including measures of emotion regulation and depression.

Measures

   PTSD Checklist-Civilian Version (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1993). The PCL-C measures the seventeen PTSD symptoms that correspond to the PTSD diagnostic criteria set forth in the DSM-IV (APA, 1994). In the current study, the PCL-C was administered over the telephone as a screening measure. Items are assessed on a five point Likert scale ranging from one (not at all) to five (extremely). In order to be eligible for the study, one partner must have a score of at least 44 on the PCL-C if the trauma is not combat related, as a psychometric analysis of the PCL-C has shown this to be an appropriate cut point (Blanchard, Jones-Alexander, Buckley & Forneris, 1996; Ruggiero, Del Ben, Scotti & Rabalais, 2003). The PCL-C has demonstrated good convergent validity (kappa = .64 with PTSD diagnosis from a

structured clinical interview; Blanchard et al., 1996) and excellent internal consistency (.94–.97; Blanchard et al., 1996).

Traumatic Life Events Questionnaire (TLEQ; Kubany, Leisen, Kaplan, Watson, Haynes & Owens, 2000). The TLEQ was used as a reference for determining participants' primary traumatic event for which symptoms were assessed during the Clinician Administered PTSD Scale (CAPS; see description below). Assessors conducting the CAPS used the TLEQ as a reference and as a discussion point to determine with the participant the most distressing traumatic event. The TLEQ measures a wide array of traumas, as it specifically queries for each trauma type, which the CAPS does not do. The TLEQ is a self-report measure that asks about the occurrence of 22 different types of trauma (e.g., military combat, sexual assault). Participants indicate the frequency that each type of trauma has occurred in their lifetime using the scale "never," "once," "twice," "three times," "four times," "five times," and "more than five times". The TLEQ also assesses for Criterion A status of the trauma, querying about fear, helplessness, and horror, as well as which trauma currently causes the most distress. The TLEQ has demonstrated moderate levels of test-retest reliability and good content validity (Kubany et al., 2000).

Clinician Administered PTSD Scale (CAPS, Blake et al., 1995). The CAPS is a 30-item interview measure that assesses DSM-IV Criterion A status, PTSD symptom severity, and PTSD diagnosis. The core symptoms of PTSD (i.e., reexperiencing, avoidance, and hyperarousal symptoms) are primarily assessed. Associated features (i.e., guilt and dissociation symptoms) are assessed as a supplement to the core symptoms. The presence of current PTSD symptoms is assessed using 17 items that correspond to DSM-IV symptoms for PTSD (i.e., five re-experiencing, seven avoidance, and five hyperarousal symptoms). Symptoms are assessed for the

previous month, and are rated by the clinician on five-point Likert scales for frequency and intensity. For example, in terms of frequency, a sample item reads, "Have you ever suddenly acted or felt as if [the event] were happening again? Tell me more about that. How often has that happened in the last month?" with response options of "never (0)," "once or twice (1)," "once or twice a week (2)," "several times a week (3)," and "daily or almost every day (4)." The same question, in terms of intensity, reads, "How much did it seem as if [the event] were happening again? How long did it last? What did people do while this was happening?" with response options of "no reliving (0)," "mild, somewhat more realistic than thinking about the event (1)," "moderate, definite but transient dissociative quality, still very aware of surroundings, daydreaming quality (2)," "severe, strongly dissociative (reports images, sounds, or smells) but retained some awareness of surroundings (3)," and "extreme, complete dissociation (flashback), no awareness of surroundings, may be unresponsive, possible amnesia for the episode (blackout) (4)." Onset and duration of symptoms are assessed, as well as subjective distress and impairment in areas such as relationships and occupation. Total symptom severity is obtained by summing both the frequency and intensity items, with higher scores reflecting greater symptom severity. Positive PTSD diagnostic status was given when individuals scored a minimum of one in frequency and two on intensity for one reexperiencing symptom, three avoidance symptoms, and two hyperarousal symptoms. Individuals also must have experienced a Criterion A event and have experienced symptoms for a minimum of one month. Individuals also needed to demonstrate subjective distress or impairment in social or occupational functioning. Subscale scores are obtained by summing reexperiencing, avoidance, and arousal items, respectively, for both frequency and intensity. While the CAPS is an interview measure, it goes beyond self-report methodology, as the clinician uses clinical judgment when doing the assessment, and does

not rely solely on the participant's verbal response (i.e., the clinician uses contextual information, behavioral observation, etc. in order to make ratings, which may or may not match the participant's verbal report). The CAPS has been shown to have high interrater reliability (.92-.99), internal consistency (.73-.85) and convergent validity with other PTSD measures (Blake et al., 1990; for a review, see Weathers, Keane & Davidson, 2001). The internal consistency of the CAPS in the current study was good ($\alpha = .89$).

Beck Depression Inventory-II (BDI-II, Beck, Steer & Brown, 1996). The BDI-II is a 21-item self-report instrument that measures symptoms of depression. Items are rated on a 4-point Likert scale, from zero to three (e.g., "I do not feel sad (*0*)" to "I am so sad or unhappy that I can't stand it (*3*)"). Items are summed with total scores ranging from zero to 63, with higher scores indicating more severe depressive symptomatology. The BDI-II has been shown to have good internal consistency, ($\alpha = .91$; Beck, Steer, Ball & Ranieri, 1996) as well as convergent validity (.93; Dozois, Dobson & Ahnberg, 1998), as measured by the original Beck Depression Inventory (Beck, Rush, Shaw & Emery, 1979; Beck et al., 1961). The internal consistency of the BDI in the current study was good ($\alpha = .86$).

Emotion Regulation Questionnaire (ERQ, Gross & John, 2003). The ERQ is a ten item self-report measure developed to assess the use of two emotion regulation strategies: cognitive reappraisal and expressive suppression. Item examples include "When I want to feel more positive emotion (such as joy or amusement), I change what I'm thinking about" (cognitive reappraisal), and "I control my emotions by not expressing them" (expressive suppression). Items are rated on a 7-point Likert scale (1 = *strongly disagree to* 7 = *strongly agree*). The ERQ has been shown to have adequate test-retest reliability (.69) and adequate internal consistently (.79 reappraisal, .73 suppression). The expressive suppression subscale has been shown to be

negatively related to the attention to feelings, clarity of feelings, and mood repair subscales of the Trait-Meta Mood scale (Salovey, Mayer, Golman, Turvey, & Palfai, 1995), as well as negative mood regulation as assessed by the Negative Mood Regulation scale (Catanzaro & Mearns, 1990), reinforcing the overall idea that expressive suppression tends to be more of a maladaptive emotion regulation strategy. The current study demonstrated good internal consistency (cognitive reappraisal subscale α = .80; expressive suppression subscale α = .76).

Ruminative Responses Scale (RRS, Nolen-Hoeksema & Morrow, 1991). The RRS is a 22-item self-report scale that assesses individuals' responses when they are feeling "sad, blue, or depressed." The scale is divided into a reflection subscale (e.g., "analyze recent events to try to understand why you are depressed"), a brooding subscale (e.g., "Think 'What am I doing to deserve this?'"), and a depression-related subscale (e.g., "Think about how alone you feel"). Due to the heavy content overlap between the depression subscale and MDD, using only the brooding and reflection subscales is a more conservative measure of rumination (Treynor, Gonzalez & Nolen-Hoeksema, 2003), and is similar to other measurements of rumination (Cox, Enns & Taylor, 2001; Roberts, Gilboa & Gotlib, 1998). The 22-items of this scale are rated on a 4-point Likert scale (1 = *never* to 4 = *always*), with total scores ranging from 22-88. The internal consistency of the RRS is good (α = .90), and has a test-retest reliability of .67 (Treynor et al., 2003). The RRS also demonstrates good discriminant validity (Roelofs, Muris, Huibers, Peeters & Arntz, 2006) from the Fear of Spiders Questionnaire (Szymanski & O'Donohue, 1995) and the Aggression Questionnaire (Buss & Perry, 1992). The internal consistency of the RRS in the current study was adequate (rumination-brooding α = .70; rumination-reflection α = .71).

Difficulties in Emotion Regulation Scale (DERS, Gratz & Roemer, 2004). The DERS is a 36-item self-report measure of individuals' difficulties managing their emotions. The scale is

divided into six subscales (i.e., nonacceptance of emotional responses, difficulties engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity). Items are measured on a five point Likert scale (1 = *almost never* to 5 = *almost always*), with higher scores indicating more difficulties regulating emotions. Examples of items include "I experience my emotions as overwhelming and out of control," and "When I'm upset, I have difficulty getting work done." The DERS has high internal consistency (α = .93), and test-retest reliability (r = .88). The DERS also has adequate convergent validity (Gratz & Roemer, 2004), as measured by the Acceptance and Action Questionnaire (i.e., a measure of experiential avoidance; Hayes, Luoma, Bond, Masuda & Lillis, 2006) and discriminant validity, as measured by the Negative Mood Regulation Scale (Catanzaro & Mearns, 1990) and the Emotional Expressivity Scale (Kring, Smith & Neale, 1994). The internal consistency of the DERS in the current study was excellent (α = .94), and was adequate within the subscales (nonacceptance of emotion α = .68; difficulties with impulse control α = .70; difficulties engaging in goal-directed behavior α = .68; lack of emotional awareness α = .76; lack of emotional clarity α = .70; lack of flexibility of emotion regulation strategies α = .65).

Analyses

Due to possible dependency in the couples' data (i.e., participants were not randomly sampled from the population since both partners in each couple were included in the study), all measures were tested for dependency in accordance with Kenny and colleagues' (Kenny, Kashy & Cook, 2006) guidelines for distinguishable members. That is, the correlation between male and female partners' scores on each measure was examined. Dependency was not significant in any of the measures used in the current study (see Table 1). Due to the nonsignificant results, the

data was analyzed using each individual's data without taking into account dependency within each couple.

Confirmatory factor analyses of the CAPS PTSD symptom clusters were conducted to compare the three and four-factor models. Confirmatory factor analyses of the BDI were conducted to compare the two and three-factor models. These analyses were carried out using Mplus (5th ed.; Muthen & Muthen, 2007). Several indices of model fit were used, including the standardized root mean squared residual (SRMR, recommended value ≤ .08; Hu & Bentler, 1999), the root mean squared error of approximation (RMSEA, recommended value ≤ .06; Hu & Bentler, 1999; Thompson, 2004) and the Comparative Fit Index (CFI, recommended value ≥ .95; Hu & Bentler, 1999; Thompson, 2004). The chi square goodness of fit test was also used, but was emphasized less than the other fit indices due to its sensitivity to sample size (Schumacker & Lomax, 2004). A chi square difference test was also used to compare the three and four-factor models of PTSD and the two and three-factor models of depression gained from the factor analyses.

Once the factors were confirmed, a series of correlations were calculated between each emotion regulation strategy and the total PTSD and depression scores, as well as each of the PTSD and depression symptom clusters gained from the factor analyses. Comparisons of selected correlations were performed in accordance with the guidelines presented by Meng, Rosenthal and Rubin (1992), which support Steiger (1980) and Dunn and Clark's (1969) original methods. A Sidak correction (Sidak, 1967) was applied in order to minimize Type I error due to the large number of comparisons made. The Sidak correction is less conservative than the Bonferroni correction, and has been recommended for minimizing familywise error in

nonorthogonal comparisons (Blakesley et al., 2009) by adjusting the p value to a slightly more

conservative level than the standard .05 value.

Chapter 3

RESULTS

Factor Structure of PTSD and Depression

The data supported the four factor model of the CAPS (with factors of reexperiencing,

behavioral avoidance, emotional numbing, and hyperarousal symptoms) above the three factor

model (with factors of reexperiencing, avoidance, and hyperarousal symptoms). For the four

factor model, the SRMR indicated a good fit (.07), as did the RMSEA (.07) and the CFI (.92).

The chi square goodness of fit test indicated a suboptimal fit [$\chi^2(113) = 178.13, p < .001$];

however, as discussed above, given the sensitivity of the test to sample size, it is possible that the

sample size influenced the results (Schumacker & Lomax, 2004). Factor loadings were generally

high (see Figure 2), with the exception of items C8 (i.e., inability to remember important aspects

of the trauma), C12 (i.e., sense of foreshortened future), and D16 (i.e., hypervigilance). The

inter-factor correlations were generally high, particularly between the reexperiencing and

behavioral avoidance clusters. The bivariate correlations between clusters, however, were

considerably lower (see Table 3). For the three factor model, the SRMR evidenced an adequate

fit (.07), while the RMSEA (.09), and the CFI (.85) evidenced a less adequate fit. The chi square

goodness of fit test also showed a less adequate fit [$\chi^2(116) = 236.67, p < .001$]. The factor

loadings for items C8, C12, and D16 improved minimally over the four factor model; however

interfactor correlations were still extremely high (see Figure 3). The chi square difference test

between the three factor and four factor models was significant [$\chi^2(3) = 58.54, p < .001$],

indicating that the four factor model provided a significantly better fit to the data than the three

factor model. A one factor model was also tested, which yielded suboptimal results with the chi

square goodness of fit test [$\chi^2(119) = 254.50, p < .001$], adequate results with the SRMR (.07),

and less adequate results with RMSEA (.10), and the CFI (.83). The chi square difference test between the one factor and four factor models was also significant [$\chi^2(5) = 76.37, p < .001$], indicating that the four factor model provided a significantly better fit to the data.

The data supported the three factor model (with factors of vegetative, cognitive, and anhedonia symptoms) of the BDI over the two factor model (anhedonia and undifferentiated depressive symptoms). The three factor model evidenced a less than adequate fit through the chi square goodness of fit test [$\chi^2(186) = 361.86, p < .001$], and a good fit through the SRMR (.07). Other indicators did not evidence an optimal fit (RMSEA = .09, CFI = .85). The factor loadings were generally high (see Figure 4). The inter-factor correlations were high as well, but the bivariate correlations between clusters were considerably lower (see Table 3). For the two factor model (see Figure 5), the chi square goodness of fit test [$\chi^2(188) = 389.67, p < .001$] demonstrated a less adequate fit, while the SRMR evidenced an adequate fit (.06). Other indicators evidenced a less adequate fit, such as the RMSEA (.10), and the CFI (.83). The chi square difference test between the two factor and three factor models was significant [$\chi^2(2) = 27.81, p < .001$], suggesting that the three factor model demonstrated a significantly better fit to the data than the two factor model. A one factor model was also tested, which yielded less adequate results with the chi square goodness of fit test [$\chi^2(189) = 412.23, p < .001$], and adequate results with the SRMR (.07). The one factor model also yielded less adequate results with the RMSEA (.10), and the CFI (.81). The chi square difference test between the one factor and three factor models was also significant [$\chi^2(3) = 50.37, p < .001$], suggesting that the three factor model provided a significantly better fit to the data.

Descriptive Statistics

Descriptive statistics for the measures used in the current study are presented in Table 2. The range on all measures, particularly the CAPS, demonstrates variability between participants. The BDI mean is on the upper bound of the mild depression range, and the CAPS mean is indicative of a sample of individuals who are subthreshold in terms of a categorical PTSD diagnosis. The means suggest that this sample is less severe than might be seen in a clinical population; however, this is consistent with the expectations for a mixed clinic and community recruited sample, including partners who were not screened into the study based on PTSD symptoms. Forty-seven individuals met diagnostic criteria for PTSD, with an additional 26 individuals meeting criteria for subthreshold PTSD (Blanchard et al., 1996; Rabe, Dorfel, Zollner, Maercker & Karl, 2006).

Bivariate Correlations and Comparisons of Correlations

The bivariate correlations between the PTSD and depression symptom clusters were all significant (see Table 3), with a high degree of similarity in terms of correlations between the symptom clusters and PTSD and depression total scores. Even the within-construct correlations among clusters were not substantially higher than the between-construct correlations. There were similarly high correlations occurring between the behavioral avoidance cluster of the CAPS and the vegetative cluster of the BDI ($r = .63$, $p < .001$) and between the hyperarousal cluster of the CAPS and the cognitive cluster of the BDI ($r = .63$, $p < .001$), as well as between the emotional numbing cluster of the CAPS and the anhedonia cluster of the BDI ($r = .62$, $p < .001$). This suggests that the overlap is more pervasive across clusters than previously thought. This does not support the hypothesis that the highest area of overlap between the disorders is between the emotional numbing cluster in PTSD and the anhedonia cluster in depression, particularly since

the three correlations mentioned above are not substantially higher than other between-construct correlations.

Neither PTSD nor depression total scores correlated significantly with the emotion regulation strategies of lack of emotional awareness, cognitive reappraisal, or expressive suppression (see Table 4). PTSD correlated significantly with the rest of the emotion regulation strategies ($r = .38-.65$, $p < .001$). Total depression scores also correlated significantly with the remaining emotion regulation strategies ($r = .34-.72$, $p < .001$). There was great similarity in the strength of correlations between the emotion regulation strategies and PTSD/depression. When comparing the strength of the correlations, no emotion regulation strategies that evidenced significant bivariate correlations correlated more strongly with either the PTSD or depression total scores ($z = .26-1.5$, *ns*; see Table 5), thus supporting the shared constructs argument. Although expressive suppression correlated significantly more strongly with total CAPS score than with total BDI score ($z = 2.78$, $p < .01$), it is not considered meaningful since expressive suppression did not correlate significantly with either the PTSD or depression total scores.

If the distinct constructs argument is valid, nonacceptance of emotions would be expected to correlate more strongly with PTSD, particularly the emotional numbing cluster, than depression. As indicated above, nonacceptance of emotion did not correlate more strongly with the PTSD total score than the depression total score. In addition, nonacceptance of emotion was not significantly more strongly correlated with the CAPS emotional numbing cluster than the BDI total score or the BDI vegetative, anhedonia, and cognitive clusters (see Table 6). Furthermore, although nonacceptance of emotions was somewhat more strongly correlated with the CAPS emotional numbing cluster than the other PTSD symptom clusters, these differences were not statistically significant ($z = .57 - 1.71$, *ns*).

If the distinct constructs argument is valid, impulse control difficulties would be expected to correlate more strongly with PTSD, specifically the hyperarousal cluster, than depression. As indicated above, impulse control difficulties did not correlate more strongly with the PTSD total score than the depression total score. In addition, impulse control difficulties were not significantly more strongly correlated with the CAPS hyperarousal cluster than the BDI total score or the BDI vegetative, anhedonia, and cognitive clusters (see Table 6). Furthermore, although impulse control difficulties were somewhat more strongly correlated with the CAPS hyperarousal cluster than the other PTSD symptom clusters these differences were not statistically significant ($z = .29 - 1.91$, *ns*).

If the distinct constructs argument is valid, difficulties with goal-directed behavior would be expected to correlate more strongly with depression, specifically the vegetative cluster, than PTSD. As indicated above, difficulties with goal-directed behavior did not correlate more strongly with the depression total score than the PTSD total score. Difficulties with goal-directed behavior were not significantly more strongly correlated with the BDI vegetative cluster than the CAPS total score or the CAPS reexperiencing, behavioral avoidance, emotional numbing, or hyperarousal clusters (see Table 7). Furthermore, difficulties with goal-directed behavior were not more strongly correlated with the BDI vegetative cluster than the other BDI symptom clusters ($z = .40 - 1.59$, *ns*).

If the distinct constructs argument is valid, lack of emotional awareness would be expected to correlate more strongly with depression, specifically the anhedonia cluster, than PTSD. As indicated above, lack of emotional awareness did not correlate more strongly with the depression total score than the PTSD total score. In addition, lack of emotional awareness was not significantly more strongly correlated with the BDI anhedonia cluster than the CAPS total

score or the CAPS reexperiencing, behavioral avoidance, emotional numbing, or hyperarousal clusters (see Table 7). Furthermore, lack of emotional awareness was not more strongly correlated with the BDI anhedonia cluster than the other BDI symptom clusters ($z = .84 - 2.64$, *ns*). The cognitive cluster approached a significantly higher correlation with lack of emotional awareness than the anhedonia cluster ($z = 2.64$), however, due to the nonsignificance of the bivariate correlations, this comparison is not particularly meaningful.

If the distinct constructs argument is valid, lack of emotional clarity would be expected to correlate more strongly with depression, specifically the anhedonia cluster, than PTSD. As indicated above, lack of emotional clarity did not correlate more strongly with the depression total score than the PTSD total score. In addition, lack of emotional clarity was not significantly more strongly correlated with the BDI anhedonia cluster than the CAPS total score or the CAPS reexperiencing, behavioral avoidance, emotional numbing, or hyperarousal clusters (see Table 7). Furthermore, lack of emotional clarity was not more strongly correlated with the BDI anhedonia cluster than the other BDI symptom clusters ($z = 1.09 - 2.53$, *ns*).

If the distinct constructs argument is valid, expressive suppression would be expected to correlate more strongly with depression, specifically the anhedonia cluster, than PTSD. Expressive suppression evidenced a small and statistically nonsignificant correlation with the anhedonia cluster of the BDI ($r = .10$, *ns*); however, the reexperiencing, behavioral avoidance, and hyperarousal clusters of the CAPS evidenced a similar correlation ($r = -.09 - -.15$, *ns*; see Table 4), which does not provide support for the proposed association between expressive suppression and the anhedonia cluster of the BDI. As indicated above, expressive suppression did not correlate more strongly with the depression total score than the PTSD total score. While expressive suppression was significantly more strongly correlated with the CAPS total score than

the BDI anhedonia cluster (see Table 7), none of the bivariate correlations were significant (see

Table 4). Expressive suppression was significantly more strongly correlated with the BDI

anhedonia cluster than the CAPS behavioral avoidance cluster, but again the bivariate

correlations were nonsignificant, thus this comparison is not particularly meaningful. Overall, no

meaningful conclusions can be drawn from the results of the expressive suppression analyses.

If the distinct constructs argument is valid, rumination would be expected to correlate

more strongly with depression, specifically the cognitive cluster, than PTSD. As indicated above,

rumination-brooding and rumination-reflection did not correlate more strongly with the

depression total score than the PTSD total score. The brooding subscale of the RRS correlated

strongly with the cognitive cluster of the BDI ($r = .74$, $p < .001$), and the brooding subscale was

correlated significantly more strongly with the BDI cognitive cluster than the reexperiencing,

behavioral avoidance, and hyperarousal clusters of the CAPS (see Table 7). The brooding

subscale did not differentially correlate with the cognitive cluster than the emotional numbing

cluster of the CAPS. In addition, rumination-reflection was not significantly more strongly

correlated with the BDI cognitive cluster than the CAPS total score or the CAPS reexperiencing,

behavioral avoidance, emotional numbing, or hyperarousal clusters (see Table 7). Furthermore,

rumination-brooding was more strongly correlated with the BDI cognitive cluster than the BDI

vegetative cluster ($z = 4.10$, $p < .001$), but was not more strongly correlated with the BDI

cognitive cluster than the BDI anhedonia cluster ($z = 2.39$, ns). Rumination-reflection was not

more strongly correlated with the BDI cognitive cluster than the other BDI symptom clusters ($z =$

$.96 – 2.06$, ns). The brooding subscale correlation and comparisons of correlations provides some

support for the proposed association between rumination and cognitive symptoms of depression.

Looking at the emotion regulation strategies not expected to differentially correlate between PTSD and depression, lack of flexibility of emotion regulation strategies and cognitive reappraisal did not correlate more strongly with the depression total score than the PTSD total score. All clusters were examined, given the hypothesis that these two emotion regulation strategies would not correlate more strongly with any one PTSD or depression cluster. Most of the comparisons of correlations were nonsignificant between lack of flexibility of emotion regulation strategies and the CAPS and BDI clusters (see Table 8). However, the lack of flexibility of emotion regulation strategies correlated significantly more strongly with the cognitive cluster of the BDI than the reexperiencing cluster of the CAPS. Also, none of the comparisons of correlations were significant between reappraisal and the CAPS and BDI clusters (see Table 9). These results overall provide support for the hypothesis that reappraisal and lack of flexibility of emotion regulation strategies do not differentially correlate with PTSD and depression.

Chapter 4

DISCUSSION

Overall, the results of the current study support the hypothesis that PTSD and depression within the context of trauma are shared constructs. The comparisons of correlations yielded similar results, with overwhelmingly minimal differences between PTSD and depressive symptoms in terms of emotion regulation strategies. Researchers have previously examined symptom overlap, development of each disorder over time, shared risk factors, MDD increasing the risk of PTSD and vice versa as possible explanations for the high comorbidity between PTSD and depression within the context of trauma. Across diverse methods, results tend to favor the shared constructs argument (Elhai et al., 2010; Franklin & Zimmerman, 2001; Fu et al., 2007; Hyer et al., 1999; Koenen et al., 2008, Schindel-Allon et al., 2010, Taft et al., 2009). The current study adds to this literature using yet another means of attempting to falsify the shared constructs theory.

While emotion regulation may be seen as a shared risk factor (i.e., pretrauma emotion regulation deficits may predispose an individual to PTSD and/or depression following trauma), it may also be seen as a mechanism of psychopathology (i.e., in individuals who did not have pretrauma emotion regulation deficits, maladaptive emotion regulation skills develop as a way to cope with the traumatic event). While it is currently impossible to make this distinction, it was important to examine emotion regulation in order to explore a possible underlying mechanism of the trauma response. Additionally, due to the various deficits in emotion regulation that have been found in both PTSD and depression, it was important to examine emotion regulation as a possible mechanism to inform the possible distinction between depression and PTSD within the context of trauma. There are several possible underlying mechanisms in psychopathology, such

as cognitive processes, genetic influences, and emotion regulation. In the context of better understanding PTSD, cognitive processes have been widely researched (Astin & Resick, 1998; Bremner et al., 2003; Meiser-Stedman, Dalgleish, Glucksman, Yule & Smith, 2009, Resick, Nishith, Weaver, Astin & Feuer, 2002), and genetic influences are starting to be more closely investigated (Binder et al., 2004; Caspi et al., 2003; Fu et al., 2007; Koenen et al., 2007; Koenen et al., 2008; Lee et al., 2005). However, research on emotion regulation in PTSD is more limited (Ehring & Quack, 2010; Roemer et al., 2001; Tull et al., 2007a; Tull et al., 2007b), particularly in terms of examining the issue of shared versus distinct constructs, which has not previously been examined. Emotion regulation was chosen in order to build upon and extend past research regarding the overlap between PTSD and depression within the context of trauma, and the results still supported the shared constructs argument.

The observed high correlations between the CAPS and BDI clusters (both within-constructs and between-constructs) are also important to consider in support of the shared constructs argument, as this suggests that the between-constructs clusters of symptoms that have been conceptualized as categorically distinct may be variations of the same thing. That is, PTSD symptom clusters and depression clusters may be different groupings of one construct (e.g., a "response to trauma" construct as opposed to PTSD and depression separately within the context of trauma). The high inter-cluster correlations between-constructs also suggest that the overlap between the two disorders is not simply due to overlap between anhedonia and emotional numbing, but is more pervasive across clusters, which again points to the idea of shared constructs.

Across the eleven different types of emotion regulation strategies that were examined, only two exhibited any significantly different relationships between PTSD and depression. The

first was the rumination brooding subscale. That is, although the brooding subscale of rumination did not differentially correlate with CAPS and BDI total scores, brooding did correlate significantly more strongly with the cognitive cluster of the BDI than the reexperiencing, behavioral avoidance, or hyperarousal clusters of the CAPS (but not with the emotional numbing cluster of the CAPS). This finding is consistent with previous literature associating rumination with depression symptoms (Lyubomirsky et al., 1998; Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema et al., 2008). Rumination has been examined in PTSD (Ehlers & Clark, 2000; Ehring et al., 2008) largely as a maintenance factor (i.e., trauma-related rumination maintains posttraumatic symptoms) and has been shown to predict PTSD over time (Ehring et al., 2008). Also, as rumination was developed in the context of depression, the construct and measurement of rumination may be skewed towards depression.

The second strategy to evidence a significantly different correlation between PTSD and depression was the lack of flexibility of emotion regulation strategies. That is, although lack of flexibility of emotion regulation strategies did not differentially correlate with CAPS and BDI total scores, it did correlate significantly more strongly with the cognitive cluster of the BDI than the reexperiencing cluster of the CAPS. It is noteworthy that this comparison of correlations was not significant with any of the other BDI or PTSD clusters, which suggests that the lack of flexibility of emotion regulation strategies is more similar than different across PTSD and depression within the context of trauma.

When examining overlap across the individual PTSD and depression clusters, it may not seem logical that the reexperiencing cluster of the CAPS would correlate so highly with the vegetative cluster of the BDI since reexperiencing symptoms cause distress through adding stressors (e.g., intrusive memories) and heightened emotion, while vegetative symptoms cause

distress through removal of activity and flattened emotion. However, the fact that the correlation was so high has implications for our current conceptualization of PTSD in terms of the functional nature of responses to trauma. For example, if an individual is experiencing vegetative symptoms and is unable to engage meaningfully in her/his life, this may facilitate the occurrence of reexperiencing symptoms (i.e., since the individual has little or no meaningful activity, there is more opportunity for reexperiencing symptoms to emerge). Also, vegetation may decrease the opportunity for behavioral avoidance, which may increase emotional avoidance, in turn facilitating higher reexperiencing symptoms.

The results of this study suggest that the field's current conceptualization of PTSD may be inaccurate, due to not focusing on important aspects of the disorder. Currently, we conceptualize PTSD as an anxiety disorder, and only consider the avoidance, reexperiencing, and anxiety-based hyperarousal symptoms (e.g., hypervigilance and exaggerated startle response) in the diagnosis. The results of the current study suggest that conceptualizing PTSD as an anxiety disorder may not be fully accurate (Wolf et al., 2010) and conceptualizing it partially as a mood disorder may be more accurate, which has been suggested previously (Sher, 2008). Sher (2008) supports the notion that focusing on the fear and anxiety aspects will not fully address the range of difficulties individuals with this comorbidity (i.e., symptoms from the shared constructs of PTSD and depression) experience. The current study demonstrates the importance of emotion and emotion regulation in the context of trauma, which may suggest a shift in the research to focus on the broader emotional responses following trauma (as opposed to the current focus on fear). Examining the idea of shared constructs on a broad level is critical in terms of answering the basic science question of construct validity (i.e., getting researchers in the field to consider that PTSD may be conceptually different than we previously thought).

The current study has several strengths. The community sample, along with the sample size, variability of symptomatology, and conceptualization of PTSD and depression dimensionally were methodological strengths that increase generalizeability of the study results. Another strength of the current study was the use of an interview measure (i.e., the CAPS) to estimate one construct and a self-report measure (i.e., the BDI) to estimate the other construct. Using both of these distinct methodologies should have reduced the correlation between PTSD and depression (Campbell & Fiske, 1959), yet the correlation between the two disorders remained high, which gives more support to the shared constructs idea.

A limitation of the current study was the use of multiple self-report measures, which raises an issue in terms of validity. As Tversky and Kahneman (1973) discuss, people often use general heuristics to make decisions, predictions, or assessments. Thus, some participants may have had a particular view of themselves (e.g., generally negative, whether in the context of regulating emotions or in terms of depression), which may impact how they answered certain questions or even full measures (e.g., if someone has a self-concept of being depressed, they may endorse all BDI items at a high level without stopping to think about whether each symptom is applicable). This may apply to one measure (e.g., an individual has a self-view of being depressed, yet sees him/herself as a good emotion regulator and underreports emotion regulation difficulties), or it could be pervasive to multiple measures (e.g., an individual has low self-esteem and reports difficulties on multiple measures without considering if the difficulties are actually present). Future studies should employ interview measures along with self-report measures for PTSD and depression, as well as emotion regulation. Additional methodologies may also be helpful in future studies (e.g., reports from close others).

While it appears the shared constructs argument is valid, future studies should continue to attempt to falsify this theory. While the results of the current study were consistent, rumination (brooding) did evidence a significantly higher correlation with the cognitive cluster of the BDI than the reexperiencing, behavioral avoidance, and hyperarousal clusters of the CAPS. This could be a conceptualization and measurement issue, as discussed above, but this is not clear, which indicates that further exploration of the shared versus distinct constructs issue is warranted. Also, despite the cohesive results, the field tends to see the constructs as distinct, although this seems to be shifting over time. While this question is continuing to be tested currently, there is still a lack of consensus. More data is needed to fortify the shared argument in order to make it widely accepted in the field. Other possible indicators that may evidence distinguishability of the two constructs should be tested in future studies as well. For example, given the unreliability of self-report data, it would be worthwhile to examine emotion regulation through physiological response, particularly since the results of the current study indicate that emotion regulation is important in responses to trauma. In the future, emotion regulation may be better defined in the field, and different models of emotion regulation may develop. As the field progresses, we may better be able to attempt to falsify the shared constructs theory.

Clinical Implications

The results of the current study indicate that PTSD and depression within the context of trauma are the same construct, which is important to consider in treating individuals who have experienced traumatic events. Treating either PTSD or depression primarily, with less focus on the disorder that is perceived as secondary, is not likely to be as effective for the overall symptom picture, and may take a greater amount of time. Given the aforementioned current focus on the behavioral avoidance and anxiety components of PTSD, current widely used

exposure treatments of individuals who evidence depressive symptoms may not be as effective, not only for the specific symptoms, but for the other symptoms as well. The depressive symptoms may become an obstacle for treatment of the fear and avoidance symptoms (Taylor et al., 2001). For example, the vegetative and anhedonia symptoms may make it difficult for an individual to motivate him/herself to engage in in-vivo exposure, or even to attend therapy, given the emotionally difficult nature of imaginal exposure. Given the wide variety in symptom presentation in the context of PTSD, individuals may exhibit less depressive symptoms within the shared construct of PTSD-depression. If an individual is experiencing anhedonia, depressed mood, and somatic symptoms such as sleep deprivation, exposure may be less effective than for an individual without such symptoms.

The results of the current study suggest a movement towards primary interventions targeting the acute depressive symptoms (e.g., behavioral activation; an intervention aimed toward reengaging individuals with meaningful experiences in their lives, typically used as a treatment for depression) before proceeding to more exposure-based treatments. There is preliminary evidence that behavioral activation may be helpful for those with PTSD who exhibit more depressive symptoms (Jakupcak et al., 2006; Turner & Jakupcak, 2010; Wagner, Zatzick, Ghesquiere & Jurkovich, 2007). It may be helpful, in fact, to focus on treatments that integrate aspects of depression treatment with PTSD treatment to effectively and efficiently treat those individuals who experience negative reactions to traumatic events. One such treatment is Cognitive Processing Therapy (CPT; Resick & Schnicke, 1992), in which one of the four main aspects of the treatment involves a focus on becoming more aware of emotions and thoughts. It may also be helpful to create a new treatment that addresses the added component of treating depressed mood along with the other PTSD and depressive symptoms. Based on the results of

the current study, it may be fruitful to create a treatment targeting emotion regulation deficits and examine whether it is effective for both PTSD and depression symptoms (as they are currently conceptualized). If such a treatment was similarly effective for both disorders, this would add further support to the shared constructs argument.

Considering these disorders as the same construct, within the context of trauma, also has assessment implications. For example, if an individual being assessed presents depression symptoms as primary and neglects to mention a recent traumatic event, the clinician may only assess for depression, which would miss an important part of the individual's experience and would have negative implications for treatment recommendations. Given the distinction between the two disorders in the current conceptualization, it is possible to miss an imperative piece of a client's psychopathology, either in terms of depressive symptoms or PTSD symptoms as they are currently thought of. By conceptualizing the disorders as shared constructs, clinicians are more likely to capture a fuller range of an individual's response to trauma.

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APPENDIX

Table 1

Dependency Correlations Between Male and Female Partner's Scores on Measures Used in the

Current Study

Measure	Dependency (*r*)
Clinician Administered PTSD Scale	-.16, *ns*
Beck Depression Inventory	-.05, *ns*
Difficulties in Emotion Regulation Scale (DERS) Total	-.10, *ns*
DERS-Lack of Emotional Awareness	-.14, *ns*
DERS-Lack of Emotional Clarity	.21, *ns*
DERS-Difficulties Engaging in Goal-Directed Behavior	.00, *ns*
DERS-Difficulties with Impulse Control	.08, *ns*
DERS-Nonacceptance of Emotion	-.17, *ns*
DERS-Lack of Flexibility of Emotion Regulation Strategies	.01, *ns*
Emotion Regulation Questionnaire- Cognitive Reappraisal	-.13, *ns*
Emotion Regulation Questionnaire-Expressive Suppression	-.15, *ns*
Ruminative Response Scale-Brooding	.05, *ns*
Ruminative Response Scale-Reflection	.11, *ns*

Table 2

Descriptive Statistics of Measures Used in the Current Study

Measure	Mean	SD	Range
Clinician Administered PTSD Scale (CAPS) Total	36.10	24.43	0.00-102.00
CAPS-Reexperiencing	9.91	7.98	0.00-31.00
CAPS- Behavioral Avoidance	5.96	5.43	0.00-20.00
CAPS-Emotional Numbing	7.95	7.39	0.00-26.00
CAPS-Hyperarousal	12.27	8.15	0.00-32.00
Beck Depression Inventory (BDI) Total	17.47	12.55	0.00-51.88
BDI-Vegetative	5.22	3.86	0.00-16.00
BDI-Anhedonia	5.35	4.27	0.00-17.14
BDI-Cognitive	6.82	5.86	0.00-21.00
Difficulties in Emotion Regulation Scale (DERS) Total	77.01	22.61	39.00-141.00
DERS-Lack of Emotional Awareness	14.83	4.59	6.00-25.00
DERS-Lack of Emotional Clarity	10.25	3.55	5.00-24.00
DERS-Difficulties Engaging in Goal-Directed Behavior	13.84	5.25	5.00-25.00
DERS-Difficulties with Impulse Control	11.08	4.50	6.00-25.00
DERS-Nonacceptance of Emotion	12.02	4.75	6.00-25.00
DERS-Lack of Flexibility Engaging in Regulation Strategies	16.08	6.74	8.00-37.00
Emotion Regulation Questionnaire-Cognitive Reappraisal	29.53	5.25	14.00-42.00
Emotion Regulation Questionnaire-Expressive Suppression	14.52	5.35	4.00-28.00
Ruminative Response Scale-Brooding	11.03	3.62	5.00-20.00
Ruminative Response Scale-Reflection	10.10	3.65	5.00-20.00

Table 3

Correlations Between CAPS Total Score, CAPS Clusters, BDI Total Score, and BDI Clusters

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. CAPS Total	--								
2. BDI Total	.74***	--							
3. CAPS-Reexperiencing	.87***	.61***	--						
4. CAPS-Behavioral Avoidance	.82***	.63***	.73***	--					
5. CAPS-Emotional Numbing	.82***	.66***	.57***	.53***	--				
6. CAPS-Hyperarousal	.86***	.58***	.63***	.59***	.64***	--			
7. BDI-Vegetative	.66***	.82***	.58***	.63***	.48***	.53***	--		
8. BDI-Anhedonia	.67***	.90***	.55***	.56***	.62***	.51***	.61***	--	
9. BDI-Cognitive	.65***	.94***	.53***	.51***	.52***	.63***	.59***	.64***	--

Note: ***$p < .001$. CAPS = Clinician Administered PTSD Scale; BDI = Beck Depression Inventory

Table 4

Correlations Between Emotion Regulation Strategies and CAPS and BDI Clusters

Emotion Regulation Strategy	CAPS Total	BDI Total	BDI-V	BDI-A	BDI-C	CAPS-B	CAPS-C1	CAPS-C2	CAPS-D
Nonacceptance of Emotion	.46***	.54***	.35***	.48***	.55***	.38***	.30**	.44***	.39***
Difficulties w/ Impulse Control	.44***	.34***	.37***	.30**	.30**	.29**	.33***	.41***	.43***
Goals	.45***	.37***	.35***	.29**	.38***	.27**	.29**	.48***	.45***
Lack of Emotional Awareness	.02	.05	.03	-.04	.12	-.01	.01	.03	.03
Lack of Emotional Clarity	.38***	.40***	.24**	.37***	.43***	.27**	.24**	.38***	.37***
Expressive Suppression	-.12	.09	-.11	.10	.14	-.15	-.09	-.04	-.11
Rumination-Brooding	.65***	.72***	.50***	.64***	.74***	.50***	.51***	.61***	.54***
Rumination-Reflection	.48***	.52***	.37***	.52***	.47***	.34***	.35***	.50***	.41***
Cognitive Reappraisal	-.01	-.08	-.10	-.05	-.09	.04	-.00	-.10	.02
Strategies	.52***	.56***	.43***	.53***	.55***	.34***	.35***	.53***	.55***

Notes: ** $p < .01$ ***$p < .001$. Goals = Difficulties Engaging in Goal-Directed Behavior; Strategies = Lack of Flexibility Engaging in Emotion Regulation Strategies; CAPS = Clinician Administered PTSD Scale; BDI = Beck Depression Inventory; BDI-V = BDI Vegetative; BDI-A = BDI Anhedonia; BDI-C = BDI Cognitive; CAPS-B = CAPS Reexperiencing; CAPS-C1 = CAPS Behavioral Avoidance; CAPS-C2 = CAPS Emotional Numbing; CAPS-D = CAPS Hyperarousal

Table 5

Comparisons of Correlations Between Emotion Regulation Strategies and PTSD and Depression Total Scores

Emotion Regulation Strategy	Correlation with CAPS Total (*r*)	Correlation with BDI Total (*r*)	Comparison of Correlations (*z*)
Nonacceptance of Emotion	.46***	.54***	1.37, *ns*
Difficulties with Impulse Control	.44***	.34***	1.50, *ns*
Goals	.45***	.37***	1.26, *ns*
Lack of Emotional Awareness	.02, *ns*	.05, *ns*	0.40, *ns*
Lack of Emotional Clarity	.38***	.40***	0.26, *ns*
Expressive Suppression	-.12, *ns*	.09, *ns*	2.78**
Rumination-Brooding	.65***	.72***	1.48, *ns*
Rumination-Reflection	.48***	.52***	0.65, *ns*
Cognitive Reappraisal	-.01, *ns*	-.08, *ns*	0.94, *ns*
Strategies	.52***	.56***	0.94, *ns*

Notes: ** *p* < .01 *** *p* < .001. Using the Sidak correction, z-scores ≥ 2.65 are statistically significant at *p* < .05. Goals = Difficulties Engaging in Goal-Directed Behavior; Strategies = Lack of Flexibility Engaging in Emotion Regulation Strategies; CAPS = Clinician Administered PTSD Scale; BDI = Beck Depression Inventory

Table 6

Comparisons of Correlations with Nonacceptance of Emotion and Difficulties with Impulse Control

	BDI Total	BDI-Vegetative	BDI-Anhedonia	BDI-Cognitive
	Nonacceptance of Emotion			
CAPS-Emotional Numbing	1.45, *ns*	1.10, *ns*	0.56, *ns*	1.55, *ns*
	Difficulties with Impulse Control			
CAPS-Hyperarousal	1.12, *ns*	0.79, *ns*	1.55, *ns*	1.61, *ns*

Notes: Values reflected are z-scores. Using the Sidak correction, z-scores ≥ 2.65 are statistically significant at $p < .05$. CAPS = Clinician Administered PTSD Scale; BDI = Beck Depression Inventory

Table 7

Comparisons of Correlations with Difficulties Engaging in Goal-Directed Behavior, Lack of Emotional Awareness, Lack of Emotional Clarity, Expressive Suppression, Rumination-Brooding, and Rumination-Reflection

	CAPS Total	CAPS-B	CAPS-C1	CAPS-C2	CAPS-D
	Difficulties Engaging in Goal-Directed Behavior				
BDI-Vegetative	.02, *ns*	.99, *ns*	.79, *ns*	1.54, *ns*	1.23, *ns*
	Lack of Emotional Awareness				
BDI-Anhedonia	.70, *ns*	.34, *ns*	.57, *ns*	.86, *ns*	.76, *ns*
	Lack of Emotional Clarity				
BDI-Anhedonia	.16, *ns*	1.21, *ns*	1.57, *ns*	.13, *ns*	.00, *ns*
	Expressive Suppression				
BDI-Anhedonia	2.68*	2.64, *ns*	2.02, *ns*	1.60, *ns*	2.12, *ns*
	Rumination- Brooding				
BDI-Cognitive	1.88, *ns*	3.72*	3.59*	2.36, *ns*	3.15*
	Rumination- Reflection				
BDI-Cognitive	.10, *ns*	1.59, *ns*	1.48, *ns*	.43, *ns*	.74, *ns*

Notes: Values reflected are z-scores. Using the Sidak correction, z-scores ≥ 2.65 are statistically significant at $p < .05$. BDI = Beck Depression Inventory; CAPS = Clinician Administered PTSD Scale; CAPS-B = CAPS Reexperiencing; CAPS-C1 = CAPS Behavioral avoidance; CAPS C2 = CAPS Emotional numbing; CAPS-D = CAPS Hyperarousal

Table 8

Comparisons of Correlations with Lack of Flexibility of Emotion Regulation Strategies

	1.	2.	3.	4.	5.	6.	7.
1. BDI-Vegetative	--	1.42, *ns*	1.76, *ns*	1.15, *ns*	1.09, *ns*	1.24, *ns*	1.03, *ns*
2. BDI-Anhedonia		--	.40, *ns*	2.46, *ns*	2.36, *ns*	0.00, *ns*	.26, *ns*
3. BDI-Cognitive			--	2.69*	2.57, *ns*	.60, *ns*	.54, *ns*
4. CAPS-Reexperiencing				--	.16, *ns*	2.53, *ns*	2.41, *ns*
5. CAPS-Behavioral Avoidance					--	2.31, *ns*	2.17, *ns*
6. CAPS-Emotional Numbing						--	.31, *ns*
7. CAPS-Hyperarousal							--

Notes: Values reflected are z-scores. Using the Sidak correction, z-scores ≥ 2.65 are statistically significant at $p < .05$. BDI = Beck Depression Inventory; CAPS = Clinician Administered PTSD Scale

Table 9

Comparisons of Correlations with Cognitive Reappraisal

	1.	2.	3.	4.	5.	6.	7.
1, BDI-Vegetative	- -	.56, *ns*	.12, *ns*	1.52, *ns*	1.12, *ns*	0.00, *ns*	1.23, *ns*
2. BDI-Anhedonia		--	.62, *ns*	.94, *ns*	.50, *ns*	.57, *ns*	.70, *ns*
3. BDI-Cognitive			--	1.34, *ns*	.89, *ns*	.11, *ns*	1.12, *ns*
4. CAPS-Reexperiencing				--	.59, *ns*	1.52, *ns*	.23, *ns*
5. CAPS-Behavioral Avoidance					--	1.01, *ns*	.26, *ns*
6. CAPS –Emotional Numbing						--	1.42, *ns*
7. CAPS-Hyperarousal							--

Notes: Values reflected are z-scores. Using the Sidak correction, z-scores ≥ 2.65 are statistically significant at *p* < .05. BDI = Beck Depression Inventory; CAPS = Clinician Administered PTSD Scale

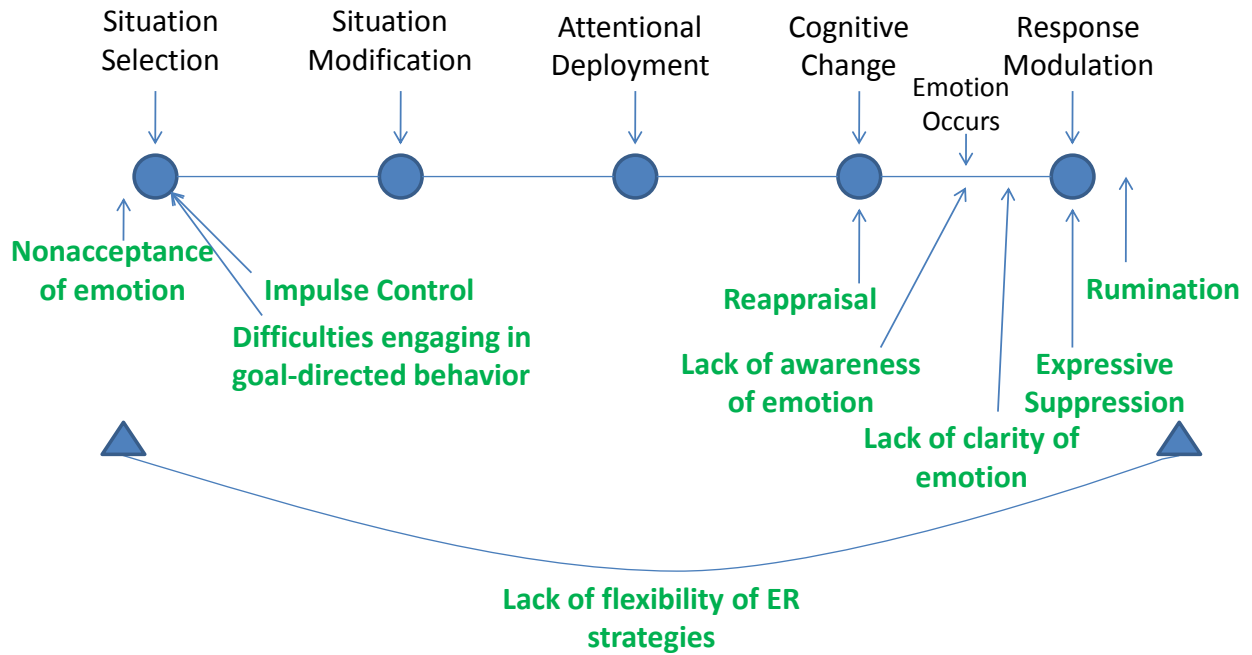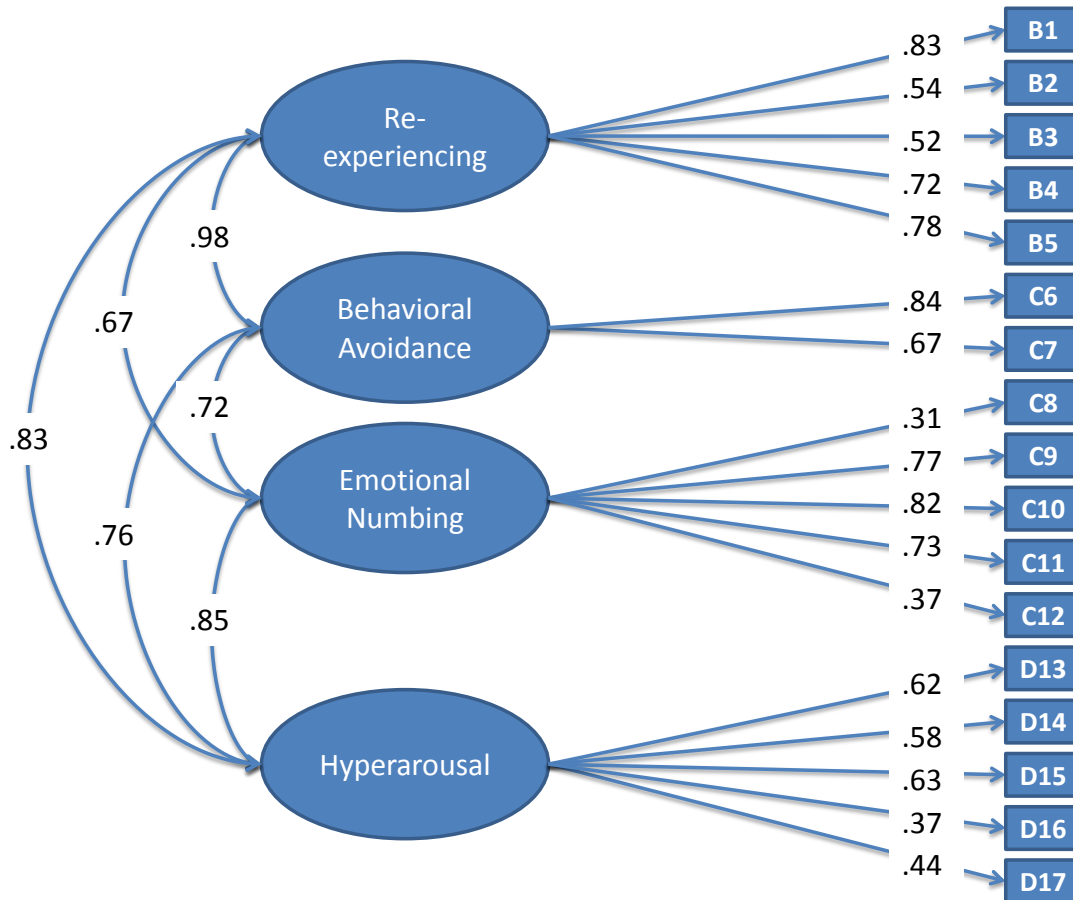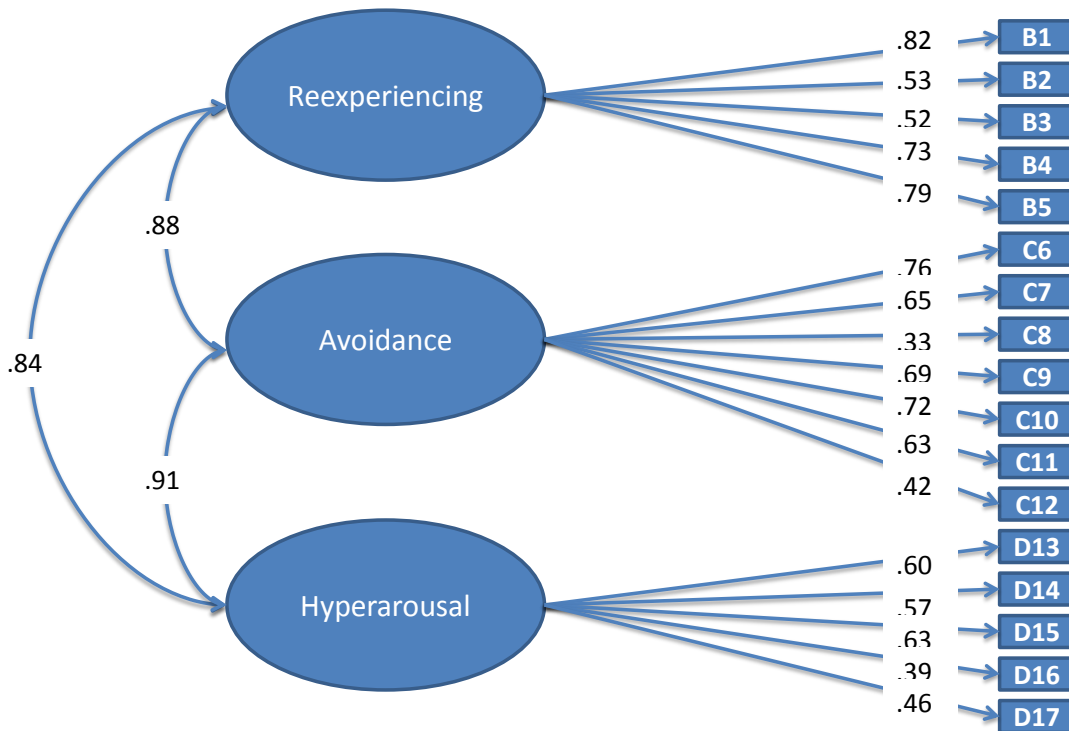Figure 1. Process Model of Emotion Regulation, from Gross and Thompson (2007)

Figure 2. Four Factor Model of the CAPS

Notes: B1-B5 = Clinician Administered PTSD Scale (CAPS) reexperiencing symptoms; C6-C12 = CAPS avoidance symptoms; D13-D17 = CAPS hyperarousal symptoms; B1 = Recurrent, intrusive images; B2 = Nightmares; B3 = Flashbacks; B4 = Emotional distress at trauma cues; B5 = Physiological reactivity; C6 = Avoidance of trauma-related thoughts, feelings and conversations; C7 = Avoidance of trauma-related people and places; C8 = Inability to recall trauma aspects; C9 = Diminished interest in activities; C10 = Feelings of detachment; C11 = Restricted range of affect; C12 = Sense of foreshortened future; D13 = Sleep difficulty; D14 =

Irritability/anger; D15 = Difficulty concentrating; D16 = Hypervigilance; D17 = Exaggerated

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Figure 3. Three Factor Model of the CAPS

Notes: B1-B5 = Clinician Administered PTSD Scale (CAPS) reexperiencing symptoms; C6-C12

= CAPS avoidance symptoms; D13-D17 = CAPS hyperarousal symptoms; B1 = Recurrent,

intrusive images; B2 = Nightmares; B3 = Flashbacks; B4 = Emotional distress at trauma cues;

B5 = Physiological reactivity; C6 = Avoidance of trauma-related thoughts, feelings and

conversations; C7 = Avoidance of trauma-related people and places; C8 = Inability to recall

trauma aspects; C9 = Diminished interest in activities; C10 = Feelings of detachment; C11 =

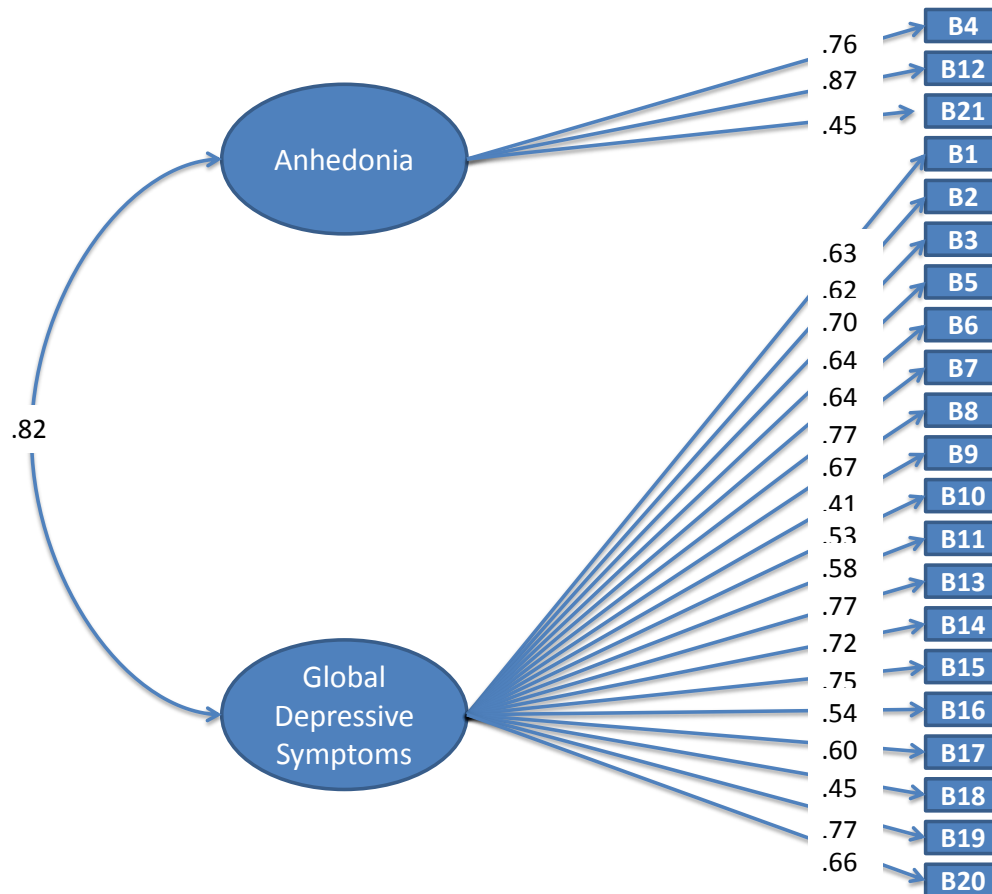Restricted range of affect; C12 = Sense of foreshortened future; D13 = Sleep difficulty; D14 =

Irritability/anger; D15 = Difficulty concentrating; D16 = Hypervigilance; D17 = Exaggerated

startle

Figure 4. Three Factor Model of the BDI
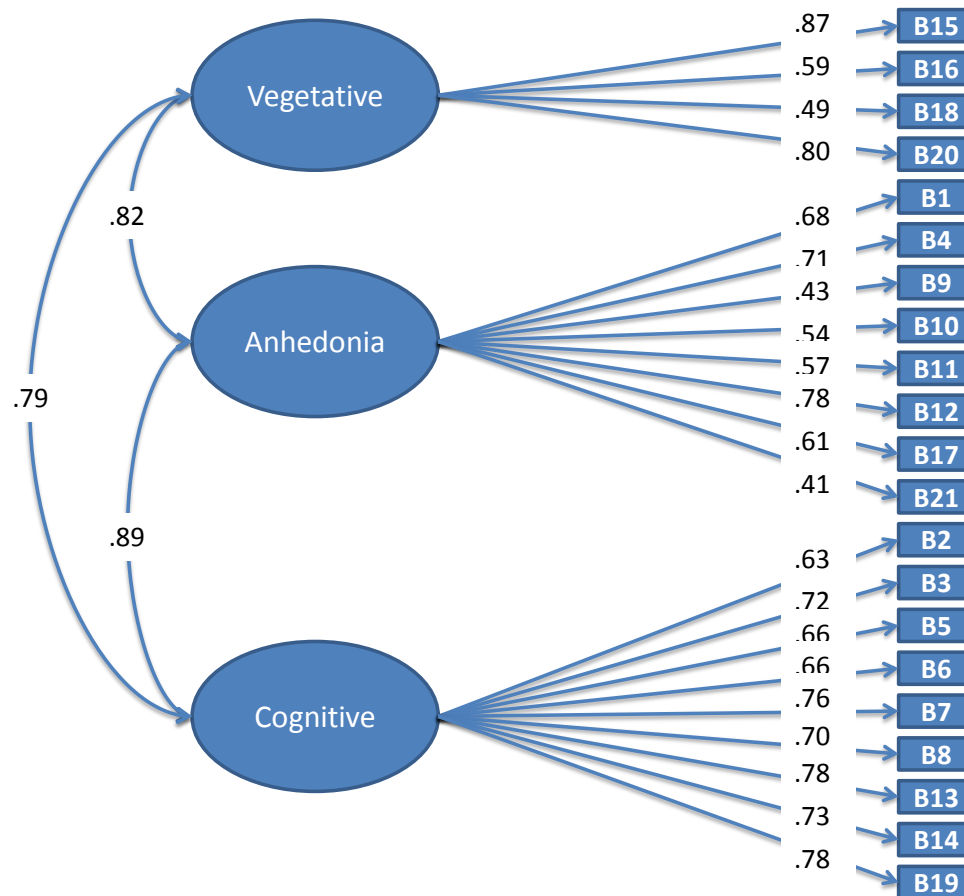
Notes: B1-B21 = Beck Depression Inventory Symptom Criteria; B1 = Feeling sad; B2 =

Discouragement about the future; B3 = Feeling like a failure; B4 = Loss of pleasure in enjoyed

things; B5 = Feelings of guilt; B6 = Feeling punished; B7 = Self-confidence; B8 = Self-criticism;

B9 = Suicidality; B10 = Crying; B11 = Restlessness; B12 = Loss of interest in people/activities;

B13 = Decision-making; B14 = Feelings of worthlessness; B15 = Energy; B16 = Sleep; B17 =

Irritability; B18 = Appetite; B19 = Concentration; B20 = Tiredness; B21 = Interest in sex

Figure 5. Two Factor Model of the BDI

Notes: B1-B21 = Beck Depression Inventory Symptom Criteria; B1 = Feeling sad; B2 =

Discouragement about the future; B3 = Feeling like a failure; B4 = Loss of pleasure in enjoyed

things; B5 = Feelings of guilt; B6 = Feeling punished; B7 = Self-confidence; B8 = Self-criticism;

B9 = Suicidality; B10 = Crying; B11 = Restlessness; B12 = Loss of interest in people/activities;

B13 = Decision-making; B14 = Feelings of worthlessness; B15 = Energy; B16 = Sleep; B17 =

Irritability; B18 = Appetite; B19 = Concentration; B20 = Tiredness; B21 = Interest in sex